Cancer is a result of a default cellular 'safe mode,' physicist proposes

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With death rates from cancer have remained largely unchanged over the past 60 years, a physicist is trying to shed more light on the disease with a very different theory of its origin that traces cancer back to the dawn of multicellularity more than a billion years ago.

In this month's special issue of *Physics World* devoted to the "physics of cancer", Paul Davies, principal investigator at Arizona State University's Center for Convergence of Physical Sciences and Cancer Biology, explains his radical new theory.

Davies was brought in to lead the centre in 2009 having almost no experience in cancer research whatsoever. With a background in theoretical physics and cosmology, he was employed to bring fresh, unbiased eyes to the underlying principles of the disease.

He has since raised questions that are rarely asked by oncologists: thinking about why cancer exists at all and what place it holds in the grand story of life on Earth.

His new theory, drawn together with Charles Lineweaver of the Australian National University, suggests that cancer is a throwback to an ancient genetic "sub-routine" where the mechanisms that usually instruct cells when to multiply and die malfunctions, thus forcing the cells to revert back to a default option that was programmed into their ancestors long ago.

"To use a computer analogy, cancer is like Windows defaulting to 'safe mode' after suffering an insult of some sort," Davies writes.

The result of this malfunction is the start of a cascade of events that we identify as cancer – a runaway proliferation of cells that form a tumour, which eventually becomes mobile itself, spreading to other parts of the body and invading and colonizing.

Orthodox explanations suppose that cancer results from an accumulation of random genetic mutations, with the cancer starting from scratch each time it manifests; however, Davies and Lineweaver believe it is caused by a set of genes that have been passed on from our very early ancestors and are "switched on" in the very early stages of an organism's life as cells differentiate into specialist forms.

The pair suggests that the genes that are involved in the early development of the embryo – and that are silenced, or switched off, thereafter – become inappropriately reactivated in the adult as a result of some sort of trigger or damage, such as chemicals, radiation or inflammation.

"Very roughly, the earlier the embryonic stage, the more basic and ancient will be the genes guiding development, and the more carefully conserved and widely distributed they will be among species," Davies writes.

Several research teams around the world are currently providing experimental evidence that shows the similarities between the expression of genes in a tumour and an embryo, adding weight to Davies and Lineweaver's theory.

Davies makes it clear that radical new thinking is needed; however, just like ageing, he states that cancer cannot generally be cured but can be

mitigated, which we can only do when we better understand the disease, and its place in the "great sweep of evolutionary history".

More information: This month's special issue of Physics World can be downloaded free of charge from 1 July 2013 at www.physicsworld.com/cws/download/jul2013

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